AMENDMENTS TO THE SPECIFICATION

Please replace paragraphs [0044] through [0049] with the following amended paragraphs [0044] through [0049]:

[0044] FIG. 11A and FIG. 11B—Determination of mini-vRNAP promoter contacts. A 20-base oligonucleotide (SEQ ID NO:30) containing wild type promoter P2 sequence binds with a 1 nM Kd (FIG. 11A). Most oligonucleotides substituted with 5-Iodo-dU at specific positions showed close to wild type affinity except for the oligonucleotides substituted at positions –11 (at the center of the loop) and –8, indicating that these positions are essential for promoter recognition (FIG. 11B). UV crosslinking indicates that mini-vRNAP primarily contacts the –11 position.

[0045] FIG. 12—Binding affinities of stem-length promoter mutants. Wild type promoter P2 with a 5bp stem has a Kd of 1 nM (top) (SEQ ID NO:31). The stem was shortened by removal of 3' bases (left) (SEQ ID NOS:32-35). The stem can be shortened by two base pairs without change in the binding affinity. The effect of lengthening the stem by addition of 3' bases is shown (right) (SEQ ID NOS:36-39). The stem can be lengthened by two base pairs without change in the binding affinity.

[0046] FIG. 13A and FIG. 13B—Identification of the transcription start site by catalytic autolabeling. A series of templates were constructed with a single C placed at different distances from the center of the hairpin (position –11) by addition or deletion of the tract of As present at promoter P2 (FIG. 13A) (SEQ ID NO:31). The affinity of mini-vRNAP for these promoters was measured by filter binding, and transcription initiation was measured by catalytic autolabeling of mini-vRNAP. All templates showed similar binding affinities. However, only the template with a C positioned 12 bases downstream from the center of the hairpin was able to support transcription initiation (FIG. 13B).

[0047] FIG. 14--UV crosslinking of mutant mini-vRNAPases to promoter oligonucleotides (SEQ ID NOS:40-41). Two mutants (K670A and Y678F) were tested for their ability to bind to wild type promoters. Both mutant RNA polymerases bound to promoter DNA with wild type affinities and crosslinked to 5-Iodo-dU substituted P2 DNA templates at positions -11 and +3 as well as the wild type enzyme, indicating that these polymerase mutations do not affect promoter binding.

[0048] FIG. 15--Run-off transcription by mutant mini-vRNAPases (SEQ ID NOS:40-41). The wild type and Y678F (SEQ ID NO:8) enzymes displayed similar activities at both template excess and template-limiting conditions, while the K670A enzyme exhibited decreased activity under both conditions. Under limiting template conditions, all three enzymes were activated by EcoSSB (right panel). However, the Y678F enzyme showed reduced discrimination between incorporation of ribo- and deoxyribonucleoside triphosphates.

[0049] FIG. 16—Mutant mini-vRNAPases in transcription initiation (SEQ ID NOS:40-41). The initiation properties of the three enzymes were compared using catalytic autolabeling. The K670A enzyme displays significantly reduced activity with the GTP derivative. The Y678F enzyme, in contrast to wild type polymerase, incorporates dATP as efficiently as rATP in a single round of phosphodiester bond formation.

Please insert the attached Sequence Listing into the specification after the abstract.